inflamed and thrombosed, and for such I believe the best treatment is an incision and removal of the clots.

2. *Internal*, which do not prolapse, and which give rise to symptoms of haemorrhage and a dragging pain. The operation for this condition is severe—though not usually accompanied by danger—and painful, and I have found that the large majority of such cases can be cured by applications of the high frequency vacuum tubes, aided by suppositories containing adrenaline.

3. *Intero-external*, which prolapse and become “nipped” by the sphincter, giving rise to great pain, and, frequently, both haemorrhage and discharge. These, and true internal haemorrhoids, are usually dilatations at the junction of the superior and middle haemorrhoidal veins.

It is for the last named very common type of pile that patients usually apply for relief, and for which sedative and astringent ointments and suppositories afford little permanent benefit.

I am convinced that the treatment by electrolysis is the most certain and the least painful. No general anaesthetic is required, and the patient is not confined to bed, as a rule, for more than twenty-four hours at most. There is no pain on defaecation after the treatment, nor reflex difficulty in emptying the bladder, and the cure, as far as the haemorrhoid treated is concerned, is permanent. The prolapsed pile is treated in the manner to be described, and the patient then left alone for ten to fourteen days. If at the end of that time there is another haemorrhoid in evidence it can be treated in a similar manner, but I have never had to administer more than three treatments in any case, and this only on one occasion.

Method.

The patient, having had the bowels thoroughly cleared out by an aperient over-night and a saline in the morning, is placed on the left side, and a large indifferent electrode, connected to the negative terminal of a source of constant current, is applied over the right buttock and hip, taking care that it is in close apposition with the skin. If the haemorrhoid is not fully prolapsed the patient is asked to strain to bring it down. The pile is now painted with a mixture of phenol gr. 30, menthol gr. 30, quinine hydrochlor. gr. 22, adrenaline gr. 1/100, which I have found to be the best “surface anaesthetic,” and after a minute or two it will be found that a local anaesthetic can be injected into the tissue below the base of the haemorrhoid without pain, provided that a sharp fine needle be employed. I usually employ sol. urea and quin. hydrochlor. (Parke, Davis and Co.). After about ten minutes the pile is completely anaesthetic, though almost immediately after being painted it can be painlessly grasped with artery catch forceps to prevent its retraction. Two or three long zinc needles, or thin spears made from “slivers” of sheet zinc and soldered to flexible insulated wire, are thrust into the base of the pile parallel to its long axis, and several more into the substance of the pile. All these needles are connected to the positive pole of the source of current, and are packed round with oiled wool to prevent their causing blisters on the parts round the anus. The current is now slowly turned on till a strength equal to about 12 to 15 milliampères per needle is reached, and is allowed to flow for ten or fifteen minutes and then gradually turned off. It will be found that the needles are quite difficult to withdraw owing to the firmness of the clot formed, and it is often well to reverse the current, making the needles negative, for a couple of minutes to facilitate withdrawal.

After the treatment the pile has changed from purple red to grey. It is covered with adrenaline and chloretone ointment and pushed well up into the rectum, and a suppository of chloretone gr. 5, morphine gr. $\frac{1}{4}$, and novocain gr. $\frac{1}{4}$ inserted. The patient is sent home with directions to go to bed for twenty-four hours, to take a dose of confection of senna and sulphur the following night, and is also instructed that, should the pile prolapse, it must be replaced at once.

The process by which the haemorrhoid is cured is twofold. The vasa vasorum are obliterated, and thus the blood supply is cut off from the walls of the vein, and at the same time a firm, closely-adherent clot is formed in the lumen of the dilated portion.

Advantages of the Method.

1. It is practically painless at the time of treatment.
2. There is very little, if any, pain afterwards.
3. The patient is not kept away from his work.

4. There is no risk of separation of the clot as far as my experience goes, or in the experience of others who have used this treatment.

5. There is no necessity to dilate the sphincter.

I do not consider this treatment is indicated in cases where the piles do not protrude, nor when they are very large.

I do not propose to quote a series of cases, but two recent ones are typical:

A lady, 75 years old, had for two years suffered from small prolapsing haemorrhoids, constant discharge from the anus which stained her linen, and intense pruritus. Two treatments at an interval of ten days, which caused her absolutely no pain either at the time or afterwards, has effected a complete cure of the haemorrhoids, of the discharge, and of the pruritus.

A medical man in busy general practice who had had a prolapsing pile for eighteen months, and discharge and pruritus, was treated on a Saturday afternoon. He had some pain that night, but visited his patients on Sunday and every subsequent day. He has had no trouble since that one treatment.

These results are typical of what one may expect with careful technique in suitable cases.

TUBERCULIN IN EPILEPSY.

BY

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For many years we have administered tuberculin with excellent effects to at least two hundred patients each week. These patients chiefly suffer from chronic ulcerative pulmonary tuberculosis and tuberculous adenitis, and are resident in the Consumption Sanatoria of Scotland, the Colony of Mercy for Epileptics, and the Orphan Homes of Scotland, Bridge of Weir, Renfrewshire.

Two cases particularly are interesting, and deserve to be recorded. Some textbooks¹ on the subject of tuberculin mention epilepsy as a contraindication to its administration. Our experience shows that, so far from that being so, it is a decided indication for its use, especially under two circumstances: (1) the presence of a tuberculous lesion, active or quiescent; (2) a family history of tuberculosis.

Six and a half years ago we began to give tuberculin to a patient (A. D.), who was 32 years of age, who had suffered from epilepsy of the major type since the age of 7, and who had a large mass of tuberculous glands below the right ear with several discharging sinuses; the mass was as large as a closed fist. There was a smaller mass supraclavicularly on the left side, and chronic pleurisy at the left base. No other change was made in the patient's life—the environment, work, and drug treatment were the same as had been the case for many years. We hoped by tuberculin to help the tuberculous condition, and had no thought of it benefiting the epilepsy. In the course of a year and a half the glands disappeared entirely and the pleurisy cleared up. *Pari passu* with the disappearance of the tuberculous lesions, the epileptic fits became fewer and after two and a half years ceased entirely, and the patient likewise improved mentally. When the fits had been absent for a period of three years the patient was discharged from the colony, and she took up work as a domestic servant. We have kept her under observation and under tuberculin treatment during the year, and she has remained perfectly well. For four years thus, apparently as the result of this one new factor—the administration of tuberculin—this patient has been free both from tuberculosis and from epilepsy.

Encouraged with the results of treatment in this case, about eighteen months ago we began to administer tuberculin to several other epileptics who had no clinically obvious or apparent tuberculosis, but gave a family history of tuberculosis. One of these (F. A.) has responded excellently. He was 14 years of age, and had had epilepsy for an indefinite period—the father could not tell how long. His mother had died of consumption. The fits were very severe in character, and one month no fewer than 338 were recorded—309 major and 29 minor. He was decidedly weak-minded, and was certified under the Mental Deficiency Act (1913) as a mental defective. Tuberculin was first given to him in November, 1919. For nearly fourteen months he has had no signs of epilepsy, and mentally he has also improved. The records of his case show that previously he was pugnacious and irresponsible, but during the past year he has been well behaved, and fairly industrious.

Five others, to whom tuberculin has been given for six to twelve months, show a diminution in the number and in the severity of their seizures. Three show no change. We have begun to give tuberculin to a hundred epileptic

patients, ignoring the presence either of a tuberculous lesion or of a hereditary strain. It is well known that *post-mortem* results point to the vast majority of those who attain maturity presenting tuberculous lesions at some part. It will be interesting in course of time to note the results.

The action of the tuberculin in epilepsy as demonstrated in those cases is uncertain. It may be due to its specific effect on some tuberculous growth in the neighbourhood of the cortex. It may be that epilepsy that is tuberculous in origin is of the Jacksonian type. This drug certainly does lead to the resolution of masses and of adhesions which are palpable, and such masses or adhesions may reflexly lead to the onset of epileptoid seizures.

The tuberculin used was Burroughs, Wellcome and Co.'s B.E. and P.B.E. mixed in equal parts, and it was given in doses beginning at 0.00000001 c.cm., gradually increased to 0.4 c.cm. The injections were given at intervals of seven days to begin with, and later of fourteen days. The treatment has gone on without any intermission. General reactions have been avoided, but once or twice local reactions of a rather severe type occurred when large doses of bacillary emulsion were given. On two occasions cold abscesses formed, which had to be aspirated.

REFERENCE.

¹ *Tuberculin in Diagnosis and Treatment*. Seventh edition, p. 233. (Randelier and Roepke.)

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

SAPRAEMIC GLYCOSURIA.

I HAVE read Dr. C. G. Higginson's article on supraemic glycosuria (February 26th, 1921, p. 296) with interest, as I have at present under my care a patient who developed glycosuria shortly after a carbuncle appeared on his neck.

My patient, a stout and previously healthy man of 64, consulted me some three weeks ago for a large boil on the nape of his neck. His urine contained no trace of sugar. In spite of topical applications the carbuncle spread rapidly, and I made a deep crucial incision throughout its extent; it continued to spread until it extended almost from ear to ear, with a breadth of some 3½ in. He was seen by a surgeon, who excised the whole of the affected tissue, leaving a large gaping wound.

Previous to the operation I volunteered the information that there was no glycosuria. Two days later the patient complained of thirst; the temperature was subnormal throughout. I again examined the urine, and found that it contained 17 grains of sugar per ounce. A diabetic regimen reduced the sugar to 4 grains per ounce in twenty-four hours, and in forty-eight hours it had completely disappeared, and has not been present since. After a period on strict diet the patient returned to ordinary diet; in spite of this he has had no further glycosuria.

Some two years ago I saw another patient with a tiny black patch on his great toe; he had 54 grains of sugar per ounce in his urine. Gangrene set in with great rapidity in spite of strict diet, and soon involved the whole foot; he also began to exhibit symptoms of impending coma. He fasted completely for four days, when the amount of sugar became reduced to 4 grains per ounce. He was then put for a time on a diet, gradually increased, of eggs and French beans, and the sugar soon disappeared. I eventually amputated his foot. He did well, and now gets about with an artificial limb. With a diet containing no sugar, but with a moderate amount of bread and one potato daily, his urine remains sugar-free, but on one occasion when he exceeded his limit of starchy food, sugar reappeared freely, to disappear again on resumption of previous restrictions.

Some years ago I had a case of carbuncle in a diabetic which, in spite of diabetic restrictions current at the time, continued to spread, the patient eventually dying of diabetic coma.

There would appear to be two types of cases—one in which the carbuncle or gangrene is the sequel to the diabetes, and the other in which the carbuncle or gangrene is the causative agent of a supraemic glycosuria, or perhaps coincident with and due to an intermittent glycosuria.

I shall be interested later on in estimating the sugar tolerance of my present patient, but shall prefer to wait for his complete recovery. The success of the Allen treatment, as expounded by Dr. Leyton, in my case of gangrene has made me think that such treatment might be generally useful in cases of diabetic gangrene, whether the gangrene be cause or effect.

Old Hill, Staffs.

THOMAS E. MITCHELL, M.B.

IN THE BRITISH MEDICAL JOURNAL of February 26th Dr. Charles Gaskell Higginson finishes his paper on supraemic glycosuria with the sentence:

"At present gangrene and carbuncle are the most striking and misleading agents in the causation of supraemic glycosuria, but further investigation will doubtless reveal a long series of such agents differing from each other very much in the power of causing supraemic glycosuria."

This prophecy has led me to record some observations which may throw a light upon the condition. The percentage of sugar in the blood of a healthy man six hours after food may be taken as 0.11, and although sugar may appear in the urine with this blood sugar, it is the exception rather than the rule; usually the blood sugar is above 0.18 per cent. in glycosurics. In pyrexia the blood sugar rises above the normal, and this is so well known that the appearance of glycosuria gives but little more anxiety than albuminuria in pyrexia.

Everyone who has had experience in the treatment of diabetes realizes that toxins produced by most infections modify metabolism sufficiently to lead to a return of sugar in the urine whilst the patient maintains the diet which was the optimum, and had permitted the blood sugar to fall to normal. The quantity of toxin may be small. I have seen the injection of one hundred million dead bacilli cause glycosuria in a woman who was upon a diet of only half her optimum. The reappearance of sugar in a diabetic child permitted of the correct prophecy, three days before their appearance, that she would develop the spots of chicken-pox. (The sister was convalescent from that disease.)

The supraemia of pyorrhoea alveolaris is so common and important a factor in diabetes mellitus that extirpation of the infection has often led to the disappearance of glycosuria with but an insignificant modification in diet. The evidence upon which this is based is rather lengthy. The causes of glycosuria are so numerous that any tentative explanation offered is offered with all humility. It seems to me probable that one form of diabetes mellitus is due to a deterioration of the cells of the islands of Langerhans in the pancreas. This deterioration is sometimes due to a direct infection accompanied by a slight pyrexia for a few days. Some of the cells are destroyed, some impaired, and others left normal. The impaired cells have a reduced vitality and may be paralysed or destroyed by various toxins. Chloroform and ether often destroy them and turn a mild diabetes into a severe one. A mild toxin may lead to a temporary glycosuria from which recovery may be complete. I tend to the view that supraemic glycosuria and glycosuria due to non-diabetic gangrene are simply two varieties of toxic glycosuria.

London, W.

O. LEYTON, M.D., D.Sc., F.R.C.P.

NORMAL DELIVERY AFTER TRAUMATIC RUPTURE OF UTERUS.

IN view of the interest aroused by the discussion at the Royal Society of Medicine, in May of last year, on spontaneous rupture of the uterus during labour, following Caesarean section, I think the following case, unique I believe, will be of interest.

A married woman, aged 22, five months pregnant, fell from a railway platform on to the rails, rupturing the uterus. I examined her two, seven, and eleven hours after the accident, and the uterus appeared normal. As at the last examination she showed symptoms of internal haemorrhage, I diagnosed concealed haemorrhage, and, turning her on to the left lateral position, tried to rupture the membranes. As I was unsuccessful in this, I got Dr. Gordon Ley to see the case. He saw her fifteen hours after the accident, and decided to open the abdomen. When this was done, he found the uterus ruptured on its anterior surface for about 9 inches, and the foetus and membranes lying in the abdominal cavity. I believe the foetus had slipped out of the uterus, when I put the patient into the left lateral position, eleven hours after the accident. The foetus, placenta and membranes were removed, the uterus stitched up with two layers of No. 3 chromicized catgut, the abdominal cavity swabbed out, and the abdomen closed. Nineteen months later the patient became pregnant and was delivered after a normal labour at full time. The placenta was somewhat adherent